Blood gases

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In this podcast, Josh Pillemer talks about blood gases.

"What does the gas show?" A simple question you hope nobody EVER asks you! In fact, the world of acid-base is an intimidating monster that terrifies even the most seasoned ward doctors. Even though acid-base is impossible to understand, there are some tips that can help make the interpretation of blood gases less daunting.

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About Dr Josh Pillemer

Josh Pillemer is an Intensive Care Fellow at Royal North Shore Hospital in Sydney. Although he thoroughly enjoys the excitement and stimulation of ICU medicine, he is learning that it's the quieter moments spent with patients and their families that matter the most.

Blood Gases

With Dr Josh Pillemer, Intensive Care Fellow, Royal North Shore Hospital, Sydney, New South Wales, Australia.

Introduction

Interpreting blood gases is an intimidating task for junior doctors. The best way to master this skill is to go through as many blood gases as you can in a systematic manner.

1. You have a patient's blood gas results which read: pH 7.4, pCO2 20, Bicarb 16 and BE -8

- This person has a very low CO2 (respiratory alkalosis) and bicarbonate (metabolic acidosis)
- Overall however the pH is normal
- The real question is, 'what is the underlying process causing this derangement?'

2. What is the difference between an 'osis' and an 'aemia'

- The difference between an acidosis and acidaemia and alkalosis and alkalaemia is an important place to start to avoid confusion
- An acidosis is a process that would make the blood acidotic if there were no opposing forces
- An alkalosis is a process that would make the blood alkalotic if there was no other opposing force
- Acidaemia is when the pH is less than 7.35 and alkalaemia is when the pH is more than 7.45
- So in the previous example, there is both metabolic acidosis and respiratory alkalosis, but neither acidaemia nor alkalaemia

3. ABG vs VBG

- In practical terms, in the emergency setting there is very little to gain from an arterial sample over a venous sample
- The CO_2 difference between arterial and venous sample are unlikely to vary by a degree that will change your management
  - if the pCO_2 is normal on a VBG, an ABG adds nothing
  - if the pCO_2 is grossly elevated on a VBG and the patient is unwell, you certainly wouldn't delay management waiting for an ABG
- There is little difference between arterial and venous results for pH and bicarbonate (certainly not a large enough difference to largely alter management)
  - Venous pO_2 is not helpful in answering questions about oxygenation, and advice is to ignore it. The exception here is with a mixed or central venous gas – but that is a whole different story
- So in the emergency setting, you do not need to insist on an arterial gas unless:
  - You have a specific oxygenation question (though this will not tell you much over a good saturation trace anyway)
  - Or if you do not trust the results of the venous sample

4. The four main acid base processes

- In health the pH lies between 7.35 and 7.45, so anything above or below this range are acidaemic or alkalaemic respectively
- Respiratory Acidosis = $pCO_2 > 45$
- Respiratory Alkalosis = $pCO_2 < 35$
- Metabolic Acidosis = Bicarbonate <22
- Metabolic Alkalosis = Bicarbonate >26
- Although respiratory and metabolic alkaloses are interesting, they very rarely lead to death – so we are going to focus on the acidoses

5. Respiratory Acidosis

- Causes of high CO_2 are either that you are not getting rid of it or that you are making too much
- If you are not getting rid of it that can be a central problem (e.g. Hypoventilation/drowsy/narcotised) or a physiological problem that is not letting the CO_2 get out (e.g. COPD/asthma/PE/ventilation-perfusion mismatch)
- Causes of making too much CO_2 are not as common but include increased metabolic rates/hyperthyroidism/malignant hyperthermia

6. Compensation

- There are equations that you can use to determine compensation
- In respiratory acidosis: if your CO_2 is going up your bicarb should also go up in compensation
    - For every 10 that your CO_2 goes up from normal (40), your bicarb should go up by 1 if it is an acute process or by 4 if it is a chronic process
    - For example, if you have a CO_2 of 60 (or a rise of 20 from normal), the bicarb should be between 26 (2 above normal = acute) or 32 (8 above normal = chronic)
    - If the bicarbonate is lower than this expected level, then there may be another process (namely, a metabolic acidosis) inhibiting it

7. Say you are shown a blood gas for a patient with a pH of 7.2 and pCO_2 of 68. What are you going to do with that information?

- The numbers only ever tell part of the story!
- Although this may represent an uncomplicated respiratory acidosis, it may in fact be an acute metabolic acidosis in someone who usually has a raised pCO_2 – and although a detailed history would clarify this, your first priority is to ASSESS THE PATIENT
- If this gas came from a well-looking non-distressed patient, then you have time to take a history, perform an examination, call your registrar, perform some simple therapies (bronchodilators for wheeze, steroids for COPD exacerbation, etc.) and talk to the Respiratory team if indicated
- If the patient is drowsy, tachypnoeic, hypoxaemic – or otherwise “sick” looking – then call for **immediate** assistance

- This could be either through a RRT/MET/CERS call, ICU referral, or activation of an Arrest Call (depending on the situation, and the local process of escalation)

8. Metabolic Acidosis

- There are two types of metabolic acidosis:
  - High anion gap
  - Normal anion gap
- The causes of normal metabolic acidosis are:
  - Kidneys – renal tubular acidosis, addison's disease etc
  - Gut – losing large amounts of bicarbonate eg diarrhea, small bowel fistulae
  - Iatrogenic – eg. gave too much chloride (0.9% Saline)
- Anion gap = Na^+ – (Cl^- + HCO_3^-)
  - The normal value is 12, with a range of 8-16
  - If you include Potassium in the equation then the normal value goes up by 4 to 16, as does the range to 12-20
- High anion gap metabolic acidosis can be caused by:
  - K = Ketoacids
    - Most commonly overlooked – in starvation, alcoholism as well as diabetes
  - I = Ingestion
    - Methanol, salicyclate, ethyleneglycol
  - L = Lactate
  - U = Urea
    - NB: Urea is not an anion itself, but this represents glomerular dysfunction, where the kidney is not excreting the other anions (suphates, phosphates) it usually does

9. Anion gap – Albumin correction

- A large amount of your anion gap is made up of the negative charge on albumin, which means that if albumin drops, the anion gap changes a lot. There is therefore a corrective factor for a low albumin = you take your anion gap and add a quarter of the difference from your current albumin to your normal albumin (40g/l)
- Eg if your anion gap is 12, albumin is 20 (i.e. half normal)

- Corrected anion gap is 12 + 1/4×20 = 17
- It has gone from a normal anion gap to a high anion gap

10. Delta ratio

- Things get a little complicated from here, and goes beyond the realms of your RMO role!
- A pure raised anion gap acidosis should theoretically have an equal change in the anion gap to the change in the bicarbonate (that is, for every mmoL of extra unmeasured anion generated, there will be a corresponding decrease in the serum bicarb concentration)
    - As such, when you look at the ratio of the change in anion gap to the change in bicarbonate, this ratio (the delta ratio) will be 1
- A normal gap acidosis will have a much greater change in bicarbonate from normal than change in anion gap from normal
    - As such, the ratio of the change in anion gap to change in bicarbonate will be SMALLER than 1
- Experimentally, it is suggested that a delta ratio of 1 or more points towards the presence of a pure raised anion gap metabolic acidosis, while a delta ratio of 0.4 or less points towards a pure normal gap acidosis
    - If the ratio lies between 0.4 and 1, it is likely there is a mixture of the two

11. Don't underestimate the EUC in acid-base analysis

- Even though an EUC does not give you a pH, it does give you a huge amount of information about the metabolic state
    - The bicarbonate is a good indication of overall metabolic state
    - The anion gap is calculated from Na+, Cl- and HCO3-
    - From these, you should be able to detect a clinically significant metabolic acidosis, and have some information to suggest a cause (raised vs normal gap)
- I have lost count of the number of desperately unwell people that have had a delayed recognition of critical illness because nobody paid any attention to the HCO3- on the EUC!

12. Compensation

- There are compensation rules for metabolic acidosis as well, to determine the adequacy of the respiratory response
    - Expected pCO2 = 1.5 x [HCO3-] + 8 (+/- 2)
- For example, if the patient's [HCO3-] is 12, their expected pCO2 (in the setting of full compensation) would be:
    - 5 x 12 + 8 (+/-2) = 18 + 8 (+/-2) = 26 (+/-2)
    - e. the expected pCO2 would be between 24 and 28 mmHg
- If the pCO2 is higher than this, then there is a concurrent respiratory acidosis
- If it's lower, there's likely a concurrent respiratory alkalosis

13. Conclusion

- Unfortunately, everything we have discussed is entirely oversimplified, and possibly even untrue
- All of the rules I mentioned were derived from healthy volunteers and may not be entirely accurate in unwell patients
- Furthermore, a man named Stewart did some very clever work to actually find that bicarbonate is not actually a causative factor in acid-base states – it is purely a dependent observer that changes in response to the real causative factors
    - This is a VERY complicated concept that is best left to your Primary examinations if you end up working in critical care
    - Otherwise, the rules and approaches we have discussed give a solid approach to acid-base
    - Bicarbonate is a dependent variable, cannot determine acid base alone

Useful Resources

- Anaesthesia MCQ Acid Base Book
- Blood Gases with Josh Pillemer: 2018 onthewards conference in Sydney, Australia

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